Paediatric Respiratory Reviews xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Paediatric Respiratory Reviews

Review

Dental Treatment for Paediatric Obstructive Sleep Apnea

Joachim Ngiam [a], Peter A. Cistulli [b,*]

[a] University of Sydney, Sydney, Australia. Research Fellow, Department of Respiratory and Sleep Medicine, Royal North Shore Hospital, and Sydney Medical School, University of Sydney, Sydney, Australia
[b] Centre for Sleep Health & Research, Department of Respiratory and Sleep Medicine, Royal North Shore Hospital and Sydney Medical School, University of Sydney, Sydney, Australia

EDUCATIONAL AIMS

The reader will be able to:

- Appreciate the complex interplay between normal respiration, craniofacial growth and development and its contribution to paediatric obstructive sleep apnea (OSA).
- Discuss the current evidence supporting the use of rapid maxillary expansion, oral appliances and distraction osteogenesis in the treatment of paediatric OSA.
- Define the indications and limitations for dental treatment for paediatric OSA

ARTICLE INFO

Keywords:
Obstructive sleep apnea
Rapid maxillary expansion
Oral appliances
Distraction Osteogenesis

SUMMARY

Paediatric obstructive sleep apnea (OSA) is common and its prevalence is expected to increase due to the rise in childhood obesity. Recent research has shown that many children, both syndromic and non-syndromic, who exhibit mouth breathing as a result of upper airway obstruction, may also exhibit dentofacial anomalies. Although adenotonsillectomy and continuous positive airway pressure have been classically proposed as the primary treatment modalities for paediatric OSA, there are significant limitations to both therapies. Therefore newer treatment modalities are needed. Current research has focused on emerging dental treatment options for paediatric OSA, such as rapid maxillary expansion, oral appliances and distraction osteogenesis. However, there are few randomized trials assessing the effectiveness of these novel dental therapies for paediatric OSA, and hence further research is required to advance the field.

INTRODUCTION

The obstructive sleep apnea syndrome (OSAS) in children is defined as a disorder of breathing during sleep characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction (obstructive apnea) that disrupts ventilation during sleep and fragments sleep patterns [1]. It is estimated that 3–26% of young children are habitual snorers [2–5] with 1.2% to 5.7% of the general paediatric population exhibiting OSAS [6–8]. The peak incidence has been reported to occur between the ages of 2 and 8 years old and is generally thought to be due to a discrepancy between the size of lymphoid tissue and airway calibre. OSAS is characterised by upper airway collapse during sleep due to an imbalance between upper airway structure contributed by factors such as adenotonsillar hypertrophy, craniofacial anomalies, upper airway neuromuscular tone and obesity. The sequelae of OSAS include neuropsychological and cognitive impairment, systemic [9,10] and pulmonary hypertension [11] and endothelial dysfunction [12].

Adenotonsillectomy (AT) has been generally proposed as the treatment of choice for children with paediatric OSAS. However, several studies have highlighted the multi-factorial nature of this condition with craniofacial anomalies, syndromic conditions such as Downs syndrome, obesity and OSA severity playing key factors in residual OSAS after AT intervention [13–16]. Complete resolution of OSA defined as AHI $<$ 1 event/hour has been reported to range between 25% - 40% [15,17,18]. The recent Childhood

* Corresponding author.
E-mail addresses: jongiam1@hotmail.com (J. Ngiam), peter.cistulli@sydney.edu.au (P.A. Cistulli).

http://dx.doi.org/10.1016/j.prrv.2014.11.002
1526-0542/

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

Adenotonsillectomy Trial (CHAT) has provided new evidence evaluating the efficacy of early AT versus a conservative "watchful waiting" approach in children (age range 5-9 years) with moderate OSA (AHI range 2-30 event/hr) [19]. Marcus and investigators found beneficial effects of early AT improvements in certain domains including polysomnographic outcomes and quality of life, but no significant change in attention or executive function. Nevertheless, the role of AT in milder OSAS is unclear and warrants further investigation.

Nasal continuous positive airway (nCPAP) pressure remains a non-surgical treatment alternative for paediatric OSAS. However, limited compliance to this mode of therapy remains a realistic limitation in children [20–22]. Moreover, the long term implications of nCPAP therapy with mask delivered systems in growing children is poorly understood. Several studies have documented adverse dentofacial side effects including mid-facial hypoplasia following prolonged nCPAP therapy [23–25]. However, a recent small cephalometric study in children (mean age 9.0 years) undergoing PAP for a minimum of 6 months for at least 6 hours of use showed negligible change [26]. Nevertheless there is a need for treatment alternatives that are equally effective, and preferably targeting the individual pathophysiology in each child.

The current review will discuss the relationship between craniofacial development and paediatric OSAS and focus on emerging dental treatment modalities, including rapid maxillary expansion, oral appliance therapy, and maxillo-mandibular surgical interventions such as distraction osteogenesis in the co-management of paediatric sleep disordered breathing (SDB).

CRANIOFACIAL GROWTH AND FUNCTIONAL IMPLICATIONS ON CRANIOFACIAL AND DENTAL FORM

The influence of the mode of breathing on craniofacial and dentofacial growth is widely debated and still steeped in much controversy [27,28]. It is generally accepted that cartilage is the primary determinant of craniofacial growth at the cranial base synchondroses. According to the functional matrix theory proposed by Moss and Salentijn [29], growth in the craniofacial and dentofacial complex occurs in response to functional needs and possibly in response to growth of the nasal cartilage [29]. This theory is based on the principle that normal nasal breathing promotes harmonious growth and exerts influence on the development of craniofacial structures by stimulating the associated structures of the head and neck region during mastication, swallowing and breathing [30,31]. Linder-Aronson proposed the cause and effect relationship between increased airway resistance and craniofacial disharmony or malocclusion [32]. Chronic nasal obstruction leads to mouth breathing, resulting in an anterior and lowered posture of the tongue, open-mouth posture, a lowered mandibular posture and reduced orofacial muscle tonicity. This is thought to be a compensatory mechanism in response to the decreased nasal airflow in an attempt to maintain respiration. The imbalance results in the disharmonious growth and development of the orofacial structures and may manifest as discrepancies in craniofacial and dentofacial form [32,33]. These may include maxillary constriction and retrusion, under-development of the mandible, altered head and neck posture and excessive proclination of maxillary teeth. Animal studies in Rhesus monkeys with induced nasal obstruction have documented a combination of these features including an increase in the facial height and reduction in maxillary length and width [31,34]. Solow and Kreiborg proposed the soft tissue stretch theory and postulated that mouth breathing leads to altered head posture and an altered pattern of muscle recruitment, this in turn presenting as an adverse contributory factor in craniofacial morphogenesis [35].

Mouth breathing has a multifactorial aetiology and may result from anatomical obstructions due to enlarged palatine and pharyngeal tonsils, enlarged turbinates, nasal septal deviation, nasal polyps or allergic rhinitis. Children who mouth breathe due to adenotonsillar hypertrophy commonly exhibit a forward head posture, a retrognathic mandible, an increased anterior facial height, a steep mandibular plane, and lowered position of the tongue and hyoid bone [36]. Adenoidectomy promotes a change to nasal breathing and appears to facilitate maxillary and mandibular growth [36] and normalization in incisor position [37] after 5 years. Hence, the phenomenon of mouth breathing is important as this chronic habit may adversely influence growth and development of the craniofacial and dentofacial complex.

CRANIOFACIAL AND DENTAL MORPHOLOGY IN OSA

Numerous studies have identified a range of craniofacial and dental morphological characteristics associated with OSA. These are summarised in Tables 1 and 2.

Children with obstructed breathing may exhibit craniofacial abnormalities. Lofstrand et al. [38] compared 48 obstructed children with a control group of 4-year-old children with ideal occlusion. Children who snored every night or had apnoeic episodes showed a higher rate of disturbed sleep, mouth breathing, and a history of throat infections. A smaller cranial base angle and a lower ratio of posterior/anterior total face height were also seen. The obstructed children had a narrower maxilla, a deeper palatal height, a shorter lower dental arch with a higher prevalence of lateral crossbite [38]. In a recent study, children with chronic snoring were also documented to have a dolichofacial growth pattern with high mandibular plane angle, narrow palate, and severe crowding in the maxilla and the mandible, allergies, frequent colds, and habitual mouth breathing [39]. The negative impact of respiratory obstruction is not isolated to sleep disordered breathing alone. Children with asthma also exhibit increased malocclusion and mouth breathing [40,41] with significant deviations in dento-alveolar morphology such as maxillary constriction [42].

In support of these studies, Lindsay Gray in 1975 reported similar observations in his cohort of 310 patients [43]. He proposed the use of rapid maxillary expansion (RME) for medical conditions (poor nasal airway, septal deformity, recurrent ear or nasal infection, allergic rhinitis and asthma) and dental indications (crossbite, class III malocclusion, maxillary constriction, and cleft palate). Considerable improvement in colds and respiratory

Table 1
Craniofacial morphological abnormalities in OSA

• Maxillo-mandibular retrusion in relation to anterior cranial base
• Increased mandibular plane angle
• Increased anterior facial heights
• Lowered hyoid bone
• Reduced mandibular length
• Reduced pharyngeal airway space
• Elongated soft palate
• Increased tongue size

Table 2
Dental morphological abnormalities in OSA

• Maxillary constriction
• High and narrow palate
• Open Bite
• Anterior and Posterior Crossbite
• Maxillary/mandibular dental crowding
• Decreased intermolar width

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

infection, nasal allergy and asthma were reported with over 80% of patients converting from mouth to nasal breathing. Although subjectively assessed, this early study provides a foundational basis for a paradigm shift in the management of SDB. It proposes the hypothesis that timely and early intervention may promote spontaneous correction and normalization of clinical symptoms across a broad range of dento-medical abnormalities. Not surprisingly, early treatment by either surgical or dental interventions have increasingly been investigated with key research interests focussed on the potential beneficial effects on craniofacial and dentofacial growth and development.

THERAPEUTIC IMPLICATIONS

There have been several studies supporting improvements in dentofacial morphology following surgical intervention [44–46]. Hultcrantz and colleagues reported normalization in 77% of open bites and 50-65% of buccal and anterior crossbites in 22 children treated with tonsillectomy, with better results achieved in children operated before the age of 6 years old [45]. Zettergren et al. in a five year study compared 17 OSA children with age and sex-matched controls [47]. OSA children exhibited a more posteriorly inclined mandible, a more anteriorly inclined maxilla, a greater lower anterior face height, a shorter anterior cranial base, retroclined upper and lower incisors, reduced airway space and a less pronounced nose. Five years after adenotonsillectomy, there were no significant differences between the groups except for anterior cranial base length and a shorter nose.

The results of these studies support the hypothesis that upper airway obstruction in a growing child may contribute to the evolution of OSA by promoting unfavourable skeletal and dental growth, and also highlight the possibility that earlier intervention may reverse the unfavourable effects of OSA on the craniofacial and dentofacial development.

DENTOFACIAL ORTHOPAEDICS AND PAEDIATRIC OSA

In the last 2 decades, there has been a gradual evolution of a new interdisciplinary field of dental sleep medicine. For children with SDB, recent research has focused on emerging dental treatment options for paediatric OSAS, such as rapid maxillary expansion, oral appliances and distraction osteogenesis. In both syndromic and non-syndromic children, the preliminary data have shown encouraging results and generally demonstrate some improvements in respiratory function and some alleviation of OSAS symptoms. These dental, orthodontic and surgical treatment options may soon serve as viable or adjunctive treatment alternatives to adenotonsillectomy and CPAP therapy. They play an emerging role in the management of paediatric OSAS, with the potential to normalize craniofacial and dentofacial morphology, alter tongue posture and mode of respiration, so as to restore a normal trajectory of growth and development in children.

RAPID MAXILLARY EXPANSION

Children who suffer from OSA often exhibit a narrow maxilla with a high-arched palate and maxillary hypoplasia. Maxillary hypoplasia is often defined as a maxillary transverse deficiency in comparison to the mandible. Maxillary arches of OSA subjects have been documented to be narrower, more tapered and shorter in comparison to non-snoring and non-apnoeic controls [48]. Maxillary constriction may also increase nasal resistance, reduce airflow, alter tongue posture so as to reduce posterior airway space thereby promoting OSA [49].

RME is a routine maxillary expansion technique first described by Angell in 1860, who used a jackscrew to widen the maxillary palatal halves [50]. Not surprisingly, this method of maxillary expansion was controversial when originally proposed but was brought back into favour by Haas in 1961 [51]. RME is still routinely used today as an orthodontic and orthopaedic treatment to correct maxillary transverse deficiencies and posterior crossbites. The technique consists of application of orthopaedic force to the midpalatal suture by means of a rigid device anchored to the maxillary teeth and surrounding soft tissue (see Figure 1). The maxillary and palatine bones disarticulate along the midpalatal suture with the orthopaedic forces dissipating across cranial and circum-maxillary sutures [52,53]. A triangular pattern of widening with a wider base in the anterior maxillary region has been noted to occur with significant increase in nasal width and decrease in maxillary sinus width seen with cone-beam CT (CBCT) investigations [54]. It has been suggested that RME can also improve oropharyngeal space as oral volume is increased through maxillary transverse expansion modifying the resting posture of the tongue [33,55]. In Class II patients with mandibular retrusion, forward repositioning of the tongue has also been documented to occur following RME [56–58]. It has been hypothesized that anterior tongue repositioning associated with RME could contribute to increased upper airway patency.

Several studies have documented increases in nasal width and volume [59–61] and decreases in airway resistance up to 90 days post RME [60,61]. Using computational fluid dynamics, Iwasaki et al. demonstrated significant reductions in nasal resistance and maximal negative pressure in the pharyngeal airway during inspiration [62]. These observed reductions are thought to contribute to the alleviation of paediatric OSAS. Whilst the long term effects of these changes are unclear, they have been noted to persist 11 months after RME [58] but attenuate somewhat after 30 months [61,63]. Nasal cavity and nasopharyngeal volumes have also been reported to increase significantly with CT imaging following RME [64]. In 2013, Iwasaki and co-investigators studied twenty-eight children (mean age 9.96 ± 1.21 years) with nasal obstruction who required RME treatment with CBCT imaging. Significant enlargement in pharyngeal airway volume in the RME group was observed when compared to controls [55]. However, several CBCT studies have reported significant retropalatal and not oropharyngeal airway changes in children with maxillary constriction treated with RME [65,66], suggesting that effects of RME were mainly attributed to changes in the nasal cavity.

The use of RME OSA patients was first demonstrated in adults [67,68]. Cistulli and colleagues demonstrated 70% improvement in their cohort of 10 adult patients, some of whom required surgical assistance, with significant reduction in AHI (19 ± 4 vs 7 ± 4 events/hr, $p < 0.05$) [68]. Since then there have been a growing number of studies addressing the therapeutic effectiveness of RME in children with OSAS. Pirelli and colleagues prospectively studied 31 children (mean age 8.7 years) with maxillary constriction and OSAS who did not exhibit adenotonsillar hypertrophy with an obstructive AHI (OAHI) of 12.2 ± 2.6 events /hour [69]. Four to six weeks after RME, the mean OAHI reduced to 9.8 ± 2.7 events/hour. At the 4-month follow-up, the OAHI further reduced to 0.4 ± 1.1 events/hour. The mean expansion of the maxilla was 4.32 ± 0.7 mm with a mean increase of the pyriform opening of 1.3 ± 0.3 mm also reported. A marked increase in nadir SpO_2 from $78.5 \pm 8.2\%$ to $95.3 \pm 1.7\%$ and decrease in the duration of the longest apnea time was also witnessed. In a small prospective study, Villa and investigators studied 16 OSAS patients (mean age 6.6 ± 2.0; 9 males) with dental malocclusion treated with RME [70]. At one year follow up, 14 children completed the study with two lost to follow up. Eleven children had adenotonsillar hypertrophy but did not undergo adenotonsillectomy during the study. The mean AHI decreased from 5.8 ± 6.8 events/hr to 2.7 ± 3.5 events/hr six months after RME with further reductions to 1.5 ± 1.6 events/hr ($p = 0.005$), 12 months later.

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

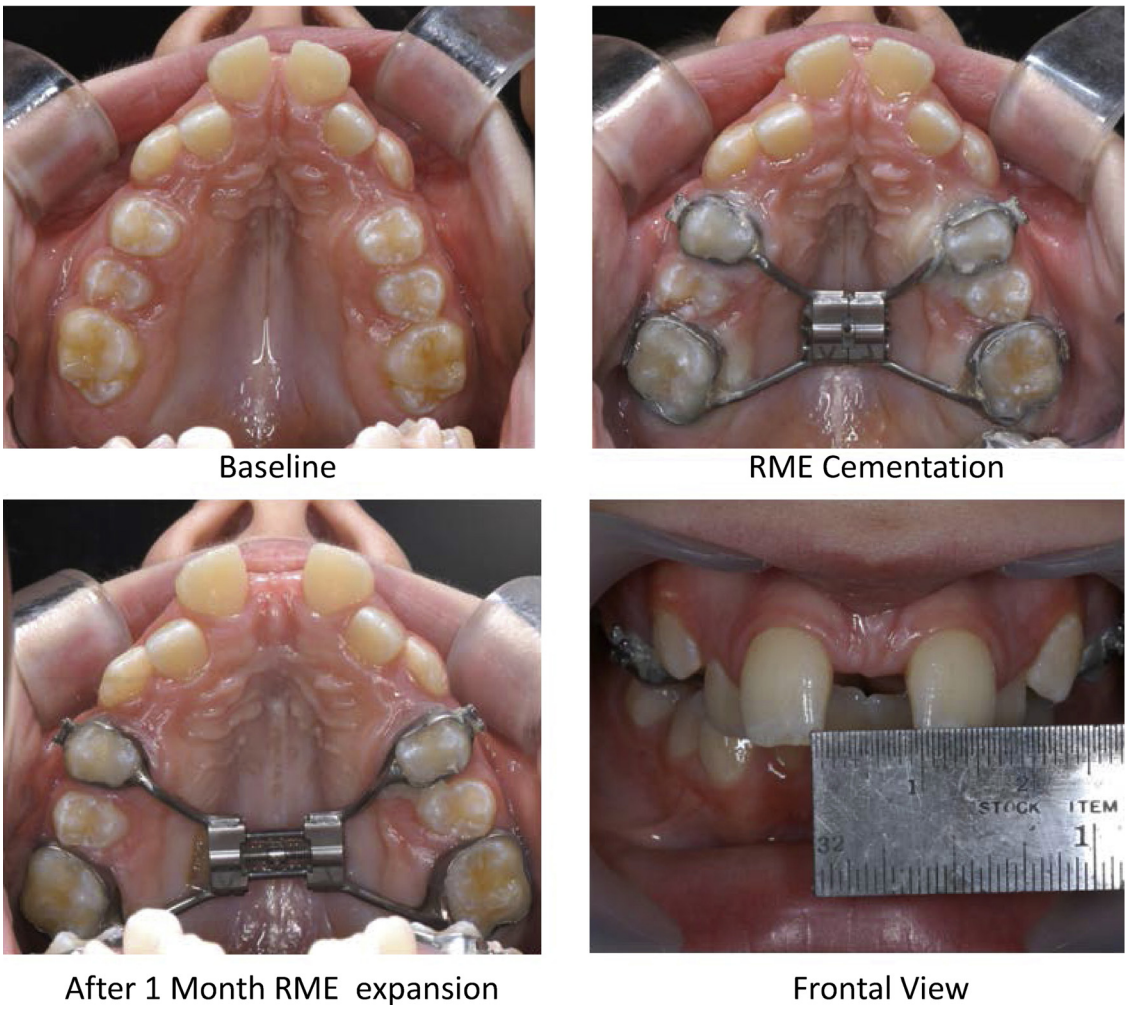

Figure 1. Photos of a child with maxillary constriction, showing fixation of the RME device to the upper molars, and the effect of active expansion on maxillary form over 1 month. This is followed a phase of retention for 6-12 months, during which the incisors migrate back to the midline and new bone formation in the midpalatine suture creates a stable structure.

The authors found that significant reductions in mean AHI occurred in children with mild tonsillar hypertrophy (5.6 to 1.0 events/hr, p = 0.034) as compared to children with severe tonsillar hypertrophy (6.2 to 2.3 events/hr, p = ns).

To study the longer term efficacy of RME, the same authors followed 8 of the 16 children initially recruited for the study [71]. Two years after completion of RME, no significant changes were noted in the mean AHI (2.4 ± 2 events/hr vs 2.3 ± 1.7 events/hr, p = NS), highlighting the potential long lasting effect of RME.

When presented with a paediatric OSAS patient, the decision as to which treatment, RME or adenotonsillectomy to perform first is unclear. In a recent study, Villa et al. investigated 52 children with OSAS [18]. Twenty-five children underwent AT (group 1) and 22 children underwent RME (group 2). Five children underwent both treatments (group 3). RME treatment was reported to be a valid treatment for group 2 with children older than 4 years old with malocclusions and mild OSA. Of note, although group 2 had a milder OSA prior to treatment when compared to group 1, a higher post-treatment AHI was found to occur (17.25 ± 13.94 events/hr to1.79 ± 1.82 events/hr, $p < 0.0001$ vs 5.81 ± 6.05 events/hr to 2.64 ± 3.11 events/hr, p = 0.005). Four of these children were noted to increase in AHI at follow up. The authors reasoned that the difference and increase in AHI in group 2 could be attributed to longer duration of the disease, obesity, allergies and potentially lower effectiveness of RME in older children. Both treatments were noted to help improve OSA, and a multidisciplinary approach with earlier RME treatment has been proposed.

MANDIBULAR ADVANCEMENT

Mandibular advancement (MA) with oral devices (OA), in particular mandibular advancement splints (MAS) in adults has been shown to be effective in treating OSA across a range of severity [72,73]. MAS are now recommended as a first line therapy for mild to moderate OSA and in more severe cases where CPAP is refused or is not able to be tolerated [74]. They enlarge the pharyngeal airway calibre predominantly at velopharynx (retropalatal) and this is mediated by an increase in its lateral dimension [75]. The improvements in airway volume are associated with soft tissue and bony structural changes. The stretching of soft tissue connections that lie within the palatoglossal and palatopharyngeal arches connecting the mandible and tongue to the soft palate and lateral pharyngeal walls has been hypothesized [76]. Chan et al. reported an increase in lower anterior facial height, raised position of the hyoid, lateral displacement of the parapharyngeal fat pads away from the airway and anterior positioning of the base of tongue muscles [75]. In a MRI study in 30 Japanese males, Baik and colleagues reported that OSA patients with retroglossal and retropalatal obstruction demonstrated a greater tendency for mandibular retrognathia, micrognathia and skeletal Class II patterns [77].

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

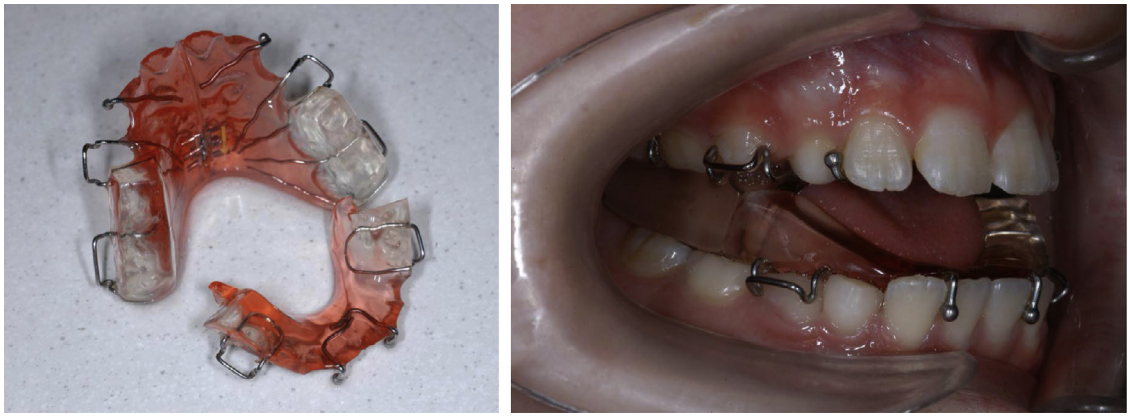

Figure 2. Photos showing an example of an oral functional appliance (twin block) used to promote mandibular growth.

In children, mandibular advancement can be attained by oral appliances (see Figure 2), distraction osteogenesis or surgical maxillo-mandibular advancement. It has been hypothesized that mandibular advancement by oral appliances (OAs) such as a functional appliance therapy may improve pharyngeal calibre size and improve the symptoms of paediatric OSAS. Children with mandibular retrognathia display a tendency towards smaller airway dimensions. An increase in posterior airway dimensions at the oropharyngeal and nasopharyngeal levels has been reported with the use of functional orthopaedic appliances in skeletal Class II children without OSA [78]. In fact, long term stability up to 22 years in pharyngeal airway size has been documented with activator-headgear treatment in children without OSA [79]. Cephalometric studies have found that children with OSA have a more retrognathic position of the mandible [77] with increased ANB angles [80] when compared with normal controls. Moreover, Matsumoto and colleagues noted that the children with an AHI $>=3$ events/hr exhibited greater mandibular retrusion when compared to a group with AHI <3 events/hr [81].

There exists a paucity of research studies on OAs in assessing their clinical effectiveness for paediatric OSAS. This is in stark contrast to adults where there exist a substantive number of published randomised controlled trials [73,82,83]. To date, there has been only one small, unblinded randomised controlled trial studying the effects of OA's in children. Villa and colleagues evaluated the clinical usefulness and tolerability of an oral jaw repositioning custom-fitted device in 32 patients (mean age, 7.1 ± 2.6 yr, 20 M) with varying orthodontic malocclusion (deep, retrusive and crossbites) [84]. Nineteen subjects were randomly assigned to an OA whereas the remaining patients acted as controls. Noteworthy, the treated group were assigned to 6 months of treatment with 24- hour use of a customised OA compared to no treatment for 6 months in the control group. Loss to follow up was 5 (26.3%) participants in treatment group and 4 (30.8%) participants in the control group. After 6 months, a reduction in the AHI from 7.1 ± 4.6 events/hr to $2.6 \pm$ events/hr ($p < 0.001$), and significant decreases in subjective reports of habitual snoring, restless leg, irritability, oral breathing and nasal stuffiness were reported. Nevertheless, only 50% of treated children achieved AHI normalisation.

In a later study, Cozza and colleagues [85] compared the effect of a modified monobloc device on 20 OSA caucasian children with ages ranging from 4 to 8 years (mean 5.91 years). The control group consisted to 20 healthy caucasian children with ages ranging from 5 to 7 years (mean 6 years). After 6 months the AHI reduced from 7.88 to 3.66 events/hr, ($p < 0.0003$).

A Cochrane database systematic review of the scientific literature to 2005 [86] assessed 384 trials and based on the above study by Villa et al. in 2002 [84], concluded that there was not sufficient evidence to state that OAs or functional orthopaedic appliances are effective in the treatment of OSAS in children. Only a recommendation that OAs or functional orthopaedic appliances may be helpful in the treatment of children with craniofacial anomalies which are risk factors for apnoea was stipulated.

Nevertheless, the evolution of new orthodontic devices continues to flourish as exemplified by the necessity to improve upper airway obstruction in infants with Pierre Robin sequence (PRS). PRS is characterised by mandibular retrognathia or retrognathia with glossoptopsis with or without cleft palate. PRS can lead to intermittent hypoxia, hypercapnia and may cause sudden death. Earlier studies have proposed the use of an intraoral orthodontic device with velar extension for intermittent upper airway obstruction [87,88]. Buchenau and colleagues in a randomised trial studied 11 infants (mean age 3 days) with PRS and compared a new orthodontic plate with velar extension compared to one without [89]. A significant change in the mean apnoea index (13.8 vs 3.9 events/hr, $p < 0.001$) with the new device was reported. A follow up 3 month study on fifteen infants (mean age 5 days) was evaluated by the same group [90]. Compared with admission (mean 17.2 events/hr; 95% CI, 11.1–26.7), there was a significant decrease in the MOAHI to discharge (mean 3.8 events/hr; 95% CI, 2.2–6.6). This improvement was sustained 3 months later (mean 1.2 events/hr; 95% CI, 0.7–2.2; p value $p < 0.001$).

In a severe paediatric OSAS case where adenotonsillar hypertrophy, nCPAP therapy and surgical treatment options were excluded, Schessl et al. reported significant improvement in a 3.5 year old boy with the use of a Frankel II functional OA [91]. Zhang et al. [92] also in a recent study evaluated the use of an oral functional appliance in children (mean age 9.7 ± 1.5 years, BMI 18.1 ± 1.04 kg/m^2) with mandibular retrognathia and OSA. 46 children (31 males 15 female), were prescribed a twin block OA for a period of 10.8 months. In contrast to adult OA treatment where unfavourable dento-alveolar changes may include bite alteration effects, treatment with functional OA in children with mandibular retrognathia in this study documented mandibular growth with accompanying dento-alveolar changes that improve the overjet, overbite and facial profile. An increase in superior posterior and middle airway space and reduction in soft palate length with two dimensional cephalometric measurements was observed. Significant improvements in AHI (14.09 ± 4.25 to 3.39 ± 1.86 events/hr, $p < 0.01$) and nadir SaO_2 ($77.8 \pm 3.38\%$ to $93.63 \pm 2.66\%$, $p < 0.01$) was documented.

DISTRACTION OSTEOGENESIS

Children born with congenital craniofacial abnormalities such as Pierre Robin sequence (PRS) or Treacher Collins syndrome are

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

faced with complex airway management issues. They often exhibit mandibular retrognathia and glossoptosis resulting in airway obstruction. Contemporary treatment options include conservative measures such as prone positioning, nasopharyngeal airway support, glossoplexy and OAs to more invasive surgical procedures such as distraction osteogenesis or tracheostomy. Mandibular distraction osteogenesis (MDO) has shown to improve upper airway obstruction and other functional outcomes [93,94], breastfeeding [95] and quality of life [96]. In a 2014 systematic review, Tahiri and colleagues evaluated the effectiveness of MDO in improving airway in the paediatric population [97]. 711 patients with a mean age of 18.1 months who underwent MDO were analysed. The patient distribution comprised of the following: isolated PRS (52.9 percent), syndromic PRS (7 percent), and Treacher Collins syndrome (6.8 percent). Success was defined as either decannulation of tracheostomy, avoidance of tracheostomy or CPAP, or alleviation or significant improvement of OSA symptoms. A staggering 89.3% of children were successfully treated with MDO. One hundred seventy-one (84.2 percent) of the 203 were successfully decannulated. 95.6% of patients with OSA experienced complete resolution or significant improvement of symptoms. The authors concluded that with proper case selection, MDO was an effective treatment procedure for the treatment of airway obstruction associated with congenital craniofacial defects involving mandibular hypoplasia.

In contrast to MDO, midface distraction osteogenesis (MFDO) is a relatively novel surgical procedure proposed for upper airway obstruction. Distinct from the mandibular lengthening and tongue base repositioning effects with MDO, the treatment goals for MFDO are aimed at the level of the nasopharynx and velopharynx. Few studies exist concerning behavioural and health and improvements with MFDO. Nevertheless, Taylor and colleagues performed a systematic review of MFDO on upper airway outcomes in 2014, [98] involving16 observational studies. MFDO was reported to improve respiratory status with favourable outcomes in cephalometry (9 studies), polysomnography (9 studies), and decannulation rates (8 studies) reported. However, only 67 patients in total were analysed by polysomnography with only mean data presented. Upper airway status was improved in most patients but long-term results and consistent objective measures were found to be lacking.

CONCLUSION

Craniofacial morphology plays a key role in the pathophysiology of paediatric OSAS. Upper airway obstruction as a result of lymphoidal tissue hypertrophy or skeletal restriction in a growing child may promote mouth breathing, impair normal craniofacial growth and development, thereby reducing pharyngeal airway size and promoting sleep-disordered breathing. Although the causative relationship between altered mode of breathing and paediatric SDB is not clearly understood, the early diagnosis and interceptive treatment of children exhibiting signs and symptoms of paediatric SDB should be encouraged.

Dental and orthodontic therapies such as RME, oral appliances and surgical treatment including distraction osteogenesis are emerging as potential treatment alternatives that may constitute an integral part in our armamentarium for paediatric OSA management. As these dental based treatment options are increasingly proposed as viable or adjunctive treatment options for a medical sleep disorder, a multidisciplinary care approach between paediatric sleep physician and dental clinician is necessary to optimise patient treatment outcomes.

Ongoing medical and dental review is required to assess treatment compliance, comfort and efficacy in the growing child. More research on their long-term clinical effectiveness, timing of treatment and patient selection for each treatment option are warranted. Future research into device specific factors, titration procedures, the prediction of treatment outcomes and the impact of growth on long- term treatment efficacy and health outcomes for paediatric OSAS is required.

CONFLICT OF INTEREST

JN and PAC do not have conflicts of interest.

RESEARCH QUESTIONS

The following questions warrant future research:

- Is the obstructive apnea hypopnea index the best metric for evaluating the response to OSAS treatment?
- In children with mild OSAS and maxillary constriction, which sequence of treatment (adenotonsillectomy or RME) should be performed first?
- What is the long term stability for RME treatment for OSAS?
- In children on long term nCPAP treatment, will a hybrid facemask reduce adverse side effects such as mid-facial hypoplasia?
- What are the phenotypes of children with OSAS that will benefit from either RME, OAs or distraction osteogenesis, or a combination of these treatment options?

References

[1] Standards and indications for cardiopulmonary sleep studies in children. American Thoracic Society. American journal of respiratory and critical care medicine, 1996; 153(2) 866–78.
[2] Ali NJ, Pitson DJ, Stradling JR. Snoring, sleep disturbance, and behaviour in 4-5 year olds. *Arch Dis Child* 1993;**68**(3):360–6.
[3] Gislason T, Benediktsdottir B. Snoring, apneic episodes, and nocturnal hypoxemia among children 6 months to 6 years old. An epidemiologic study of lower limit of prevalence. *Chest* 1995;**107**(4):963–6.
[4] Teculescu DB, et al. Snoring in French preschool children. *Pediatr Pulmonol* 1992;**13**(4):239–44.
[5] Hultcrantz E, Lofstrand-Tidestrom B, Ahlquist-Rastad J. The epidemiology of sleep related breathing disorder in children. *International journal of pediatric otorhinolaryngology* 1995;**32**(Suppl):S63–6.
[6] Bixler EO, et al. Sleep disordered breathing in children in a general population sample: prevalence and risk factors. *Sleep* 2009;**32**(6):731–6.
[7] Li AM, et al. Epidemiology of obstructive sleep apnoea syndrome in Chinese children: a two-phase community study. *Thorax* 2010;**65**(11):991–7.
[8] O'Brien LM, et al. Sleep and neurobehavioral characteristics of 5- to 7-year-old children with parentally reported symptoms of attention-deficit/hyperactivity disorder. *Pediatrics* 2003;**111**(3):554–63.
[9] Gozal D, et al. Systemic inflammation in non-obese children with obstructive sleep apnea. *Sleep medicine* 2008;**9**(3):254–9.
[10] Amin RS, et al. Twenty-four-hour ambulatory blood pressure in children with sleep-disordered breathing. *American journal of respiratory and critical care medicine* 2004;**169**(8):950–6.
[11] Miman MC, Kirazli T, Ozyurek R. Doppler echocardiography in adenotonsillar hypertrophy. *International journal of pediatric otorhinolaryngology* 2000;**54**(1):21–6.
[12] Gozal D, et al. Obstructive sleep apnea and endothelial function in school-aged nonobese children: effect of adenotonsillectomy. *Circulation* 2007;**116**(20):2307–14.
[13] Apostolidou MT, et al. Obesity and persisting sleep apnea after adenotonsillectomy in Greek children. *Chest* 2008;**134**(6):1149–55.
[14] Costa DJ, Mitchell R. Adenotonsillectomy for obstructive sleep apnea in obese children: a meta-analysis. *Otolaryngol Head Neck Surg* 2009;**140**(4):455–60.
[15] Bhattacharjee R, et al. Adenotonsillectomy outcomes in treatment of obstructive sleep apnea in children: a multicenter retrospective study. *Am J Respir Crit Care Med* 2010;**182**(5):676–83.
[16] O'Brien LM, et al. Obesity increases the risk for persisting obstructive sleep apnea after treatment in children. *International journal of pediatric otorhinolaryngology* 2006;**70**(9):1555–60.
[17] Tauman R, et al. Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. *The Journal of pediatrics* 2006;**149**(6):803–8.
[18] Villa MP, et al. Adenotonsillectomy and orthodontic therapy in pediatric obstructive sleep apnea. *Sleep & breathing Schlaf & Atmung* 2013.
[19] Marcus CL, et al. A randomized trial of adenotonsillectomy for childhood sleep apnea. *The New England journal of medicine* 2013;**368**(25):2366–76.
[20] Nixon GM, et al. Patterns of continuous positive airway pressure adherence during the first 3 months of treatment in children. *The Journal of pediatrics* 2011;**159**(5):802–7.

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

[21] Marcus CL, et al. Randomized, double-blind clinical trial of two different modes of positive airway pressure therapy on adherence and efficacy in children. *Journal of clinical sleep medicine: JCSM: official publication of the American Academy of Sleep Medicine* 2012;**8**(1):37–42.
[22] Sawyer AM, et al. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. *Sleep medicine reviews* 2011;**15**(6):343–56.
[23] Li KK, Riley RW, Guilleminault C. An unreported risk in the use of home nasal continuous positive airway pressure and home nasal ventilation in children: mid-face hypoplasia. *Chest* 2000;**117**(3):916–8.
[24] Villa MP, et al. Mid-face hypoplasia after long-term nasal ventilation. *American journal of respiratory and critical care medicine* 2002;**166**(8):1142–3.
[25] Fauroux B, et al. Facial side effects during noninvasive positive pressure ventilation in children. *Intensive care medicine* 2005;**31**(7):965–9.
[26] Korayem MM, et al. Craniofacial morphology in pediatric patients with persistent obstructive sleep apnea with or without positive airway pressure therapy: a cross-sectional cephalometric comparison with controls. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2013;**144**(1):78–85.
[27] Clark WD. Preventing dentofacial abnormalities with the proper correction of pediatric upper airway obstruction. *Archives of otolaryngology–head & neck surgery* 2005;**131**(10):916–8.
[28] Vig KW. Nasal obstruction and facial growth: the strength of evidence for clinical assumptions. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 1998;**113**(6):603–11.
[29] Moss ML, Salentijn L. The primary role of functional matrices in facial growth. *American journal of orthodontics* 1969;**55**(6):566–77.
[30] Cooper BC. Nasorespiratory function and orofacial development. *Otolaryngologic clinics of North America* 1989;**22**(2):413–41.
[31] Yamada T, et al. Influences of nasal respiratory obstruction on craniofacial growth in young Macaca fuscata monkeys. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 1997;**111**(1):38–43.
[32] Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the denition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta oto-laryngologica Supplementum* 1970;**265**:1–132.
[33] Principato JJ. Upper airway obstruction and craniofacial morphology. *Otolaryngology–head and neck surgery: official journal of American Academy of Otolaryngology-Head and Neck Surgery* 1991;**104**(6):881–90.
[34] Harvold EP, Chierici G, Vargervik K. Experiments on the development of dental malocclusions. *American journal of orthodontics* 1972;**61**(1):38–44.
[35] Solow B, Kreiborg S. Soft-tissue stretching: a possible control factor in craniofacial morphogenesis. *Scandinavian journal of dental research* 1977;**85**(6):505–7.
[36] Woodside DG, et al. Mandibular and maxillary growth after changed mode of breathing. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 1991;**100**(1):1–18.
[37] Linder-Aronson S, et al. Normalization of incisor position after adenoidectomy. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 1993;**103**(5):412–27.
[38] Lofstrand-Tidestrom B, et al. Breathing obstruction in relation to craniofacial and dental arch morphology in 4-year-old children. *Eur J Orthod* 1999;**21**(4):323–32.
[39] Huynh NT, et al. Associations between sleep-disordered breathing symptoms and facial and dental morphometry, assessed with screening examinations. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2011;**140**(6):762–70.
[40] Venetikidou A. Incidence of malocclusion in asthmatic children. *The Journal of clinical pediatric dentistry* 1993;**17**(2):89–94.
[41] Hojensgaard E, Wenzel A. Dentoalveolar morphology in children with asthma and perennial rhinitis. *European journal of orthodontics* 1987;**9**(4):265–70.
[42] Kumar SS, Nandlal B. Effects of asthma and inhalation corticosteroids on the dental arch morphology in children. *Journal of the Indian Society of Pedodontics and Preventive Dentistry* 2012;**30**(3):242–9.
[43] Gray LP. Results of 310 cases of rapid maxillary expansion selected for medical reasons. *J Laryngol Otol* 1975;**89**(6):601–14.
[44] Agren K, et al. Children with nocturnal upper airway obstruction: postoperative orthodontic and respiratory improvement. *Acta Otolaryngol* 1998;**118**(4):581–7.
[45] Hultcrantz E, et al. The influence of tonsillar obstruction and tonsillectomy on facial growth and dental arch morphology. *Int J Pediatr Otorhinolaryngol* 1991;**22**(2):125–34.
[46] Zettergren-Wijk L, Forsberg CM, Linder-Aronson S. Changes in dentofacial morphology after adeno-/tonsillectomy in young children with obstructive sleep apnoea–a 5-year follow-up study. *European journal of orthodontics* 2006;**28**(4):319–26.
[47] Zettergren-Wijk L, Forsberg CM, Linder-Aronson S. Changes in dentofacial morphology after adeno-/tonsillectomy in young children with obstructive sleep apnoea–a 5-year follow-up study. *Eur J Orthod* 2006;**28**(4):319–26.
[48] Seto BH, et al. Maxillary morphology in obstructive sleep apnoea syndrome. *Eur J Orthod* 2001;**23**(6):703–14.
[49] Warren DW, et al. The nasal airway following maxillary expansion. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 1987;**91**(2):111–6.
[50] Timms DJ. The dawn of rapid maxillary expansion. *The Angle orthodontist* 1999;**69**(3):247–50.
[51] Haas AJ. The Treatment of Maxillary Deficiency by Opening the Midpalatal Suture. *Angle Orthod* 1965;**35**:200–17.
[52] Jafari A, Shetty KS, Kumar M. Study of stress distribution and displacement of various craniofacial structures following application of transverse orthopedic forces–a three-dimensional FEM study. *The Angle orthodontist* 2003;**73**(1):12–20.
[53] Ghoneima A, et al. Effects of rapid maxillary expansion on the cranial and circummaxillary sutures. *Am J Orthod Dentofacial Orthop* 2011;**140**(4):510–9.
[54] Garrett BJ, et al. Skeletal effects to the maxilla after rapid maxillary expansion assessed with cone-beam computed tomography. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2008;**134**(1):8–9.
[55] Iwasaki T, et al. Tongue posture improvement and pharyngeal airway enlargement as secondary effects of rapid maxillary expansion: A cone-beam computed tomography study. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2013;**143**(2):235–45.
[56] Guest SS, et al. Improving Class II malocclusion as a side-effect of rapid maxillary expansion: a prospective clinical study. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2010;**138**(5):582–91.
[57] McNamara Jr JA, et al. Changes in occlusal relationships in mixed dentition patients treated with rapid maxillary expansion. A prospective clinical study. *Angle Orthod* 2010;**80**(2):230–8.
[58] Baratieri C, et al. Does rapid maxillary expansion have long-term effects on airway dimensions and breathing? *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2011;**140**(2):146–56.
[59] Cappellette Jr M, et al. Evaluation of nasal capacity before and after rapid maxillary expansion. *American journal of rhinology* 2008;**22**(1):74–7.
[60] Oliveira De Felippe NL, et al. Relationship between rapid maxillary expansion and nasal cavity size and airway resistance: short- and long-term effects. *Am J Orthod Dentofacial Orthop* 2008;**134**(3):370–82.
[61] Matsumoto MA, et al. Long-term effects of rapid maxillary expansion on nasal area and nasal airway resistance. *American journal of rhinology & allergy* 2010;**24**(2):161–5.
[62] Iwasaki T, et al. The effect of rapid maxillary expansion on pharyngeal airway pressure during inspiration evaluated using computational fluid dynamics. *International journal of pediatric otorhinolaryngology* 2014;**78**(8):1258–64.
[63] Langer MR, et al. Does rapid maxillary expansion increase nasopharyngeal space and improve nasal airway resistance? *International journal of pediatric otorhinolaryngology* 2011;**75**(1):122–5.
[64] Smith T, et al. Three-dimensional computed tomography analysis of airway volume changes after rapid maxillary expansion. *Am J Orthod Dentofacial Orthop* 2012;**141**(5):618–26.
[65] Fastuca R, Zecca PA, Caprioglio A. Role of mandibular displacement and airway size in improving breathing after rapid maxillary expansion. *Progress in orthodontics* 2014;**15**(1):p40.
[66] Zhao Y, et al. Oropharyngeal airway changes after rapid palatal expansion evaluated with cone-beam computed tomography. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2010;**137**(4 Suppl):S71–8.
[67] Palmisano RG, et al. Treatment of snoring and obstructive sleep apnoea by rapid maxillary expansion. *Australian and New Zealand journal of medicine* 1996;**26**(3):428–9.
[68] Cistulli PA, Palmisano RG, Poole MD. Treatment of obstructive sleep apnea syndrome by rapid maxillary expansion. *Sleep* 1998;**21**(8):831–5.
[69] Pirelli P, Saponara M, Guilleminault C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. *Sleep* 2004;**27**(4):761–6.
[70] Villa MP, et al. Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. *Sleep Med* 2007;**8**(2):128–34.
[71] Villa MP, et al. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. *Sleep & breathing Schlaf & Atmung* 2011;**15**(2):179–84.
[72] Ngiam J, et al. Clinical guidelines for oral appliance therapy in the treatment of snoring and obstructive sleep apnoea. *Australian dental journal* 2013;**58**(4):408–19.
[73] Sutherland K, Cistulli P. Mandibular advancement splints for the treatment of sleep apnea syndrome. *Swiss Med Wkly* 2011;**141**:w13276.
[74] Kushida CA, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. *Sleep* 2005;**28**(4):499–521.

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002

[75] Chan AS, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax* 2010;**65**(8):726–32.
[76] Isono S, et al. Anatomy of pharynx in patients with obstructive sleep apnea and in normal subjects. *J Appl Physiol* 1997;**82**(4):1319–26.
[77] Baik UB, et al. Relationship between cephalometric characteristics and obstructive sites in obstructive sleep apnea syndrome. *The Angle orthodontist* 2002;**72**(2):124–34.
[78] Ozbek MM, et al. Oropharyngeal airway dimensions and functional-orthopedic treatment in skeletal Class II cases. *The Angle orthodontist* 1998;**68**(4): 327–36.
[79] Hanggi MP, et al. Long-term changes in pharyngeal airway dimensions following activator-headgear and fixed appliance treatment. *European journal of orthodontics* 2008;**30**(6):598–605.
[80] Katyal V, et al. Craniofacial and upper airway morphology in pediatric sleep-disordered breathing: Systematic review and meta-analysis. *American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists its constituent societies and the American Board of Orthodontics* 2013;**143**(1). p. 20–30 e3.
[81] Matsumoto E, et al. Sleep architecture and the apnoea-hypopnoea index in children with obstructive-sleep apnoea syndrome. *Journal of oral rehabilitation* 2007;**34**(2):112–20.
[82] Mehta A, et al. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. *Am J Respir Crit Care Med* 2001;**163**(6): 1457–61.
[83] Gotsopoulos H, et al. Oral appliance therapy improves symptoms in obstructive sleep apnea: a randomized, controlled trial. *Am J Respir Crit Care Med* 2002;**166**(5):743–8.
[84] Villa MP, et al. Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion. *Am J Respir Crit Care Med* 2002;**165**(1):123–7.
[85] Cozza P, Ballanti F, Prete L. A modified monobloc for treatment of young children with obstructive sleep apnea. *Journal of clinical orthodontics: JCO* 2004;**38**(4):241–7.
[86] Carvalho FR, et al. Oral appliances and functional orthopaedic appliances for obstructive sleep apnoea in children. *Cochrane Database Syst Rev* 2007;(2): CD005520.
[87] Hotz M, Gnoinski W. Clefts of the secondary palate associated with the "Pierre Robin syndrome". Management by early maxillary orthopaedics. *Swedish dental journal Supplement* 1982;**15**:89–98.
[88] von Bodman A, et al. [The Tubingen palatal plate–an innovative therapeutic concept in Pierre-Robin sequence]. *Wiener klinische Wochenschrift* 2003;**115** (24):871–3.
[89] Buchenau W, et al. A randomized clinical trial of a new orthodontic appliance to improve upper airway obstruction in infants with Pierre Robin sequence. *The Journal of pediatrics* 2007;**151**(2):145–9.
[90] Bacher M, et al. An oral appliance with velar extension for treatment of obstructive sleep apnea in infants with Pierre Robin sequence. *The Cleft palate-craniofacial journal: official publication of the American Cleft Palate-Craniofacial Association* 2011;**48**(3):331–6.
[91] Schessl J, et al. Severe obstructive sleep apnea alleviated by oral appliance in a three-year-old boy. *Respiration* 2008;**76**(1):112–6.
[92] Zhang C, He H, Ngan P. Effects of twin block appliance on obstructive sleep apnea in children: a preliminary study. *Sleep & breathing Schlaf & Atmung* 2013.
[93] Hong P. A clinical narrative review of mandibular distraction osteogenesis in neonates with Pierre Robin sequence. *International journal of pediatric otorhinolaryngology* 2011;**75**(8):985–91.
[94] Ow AT, Cheung LK. Meta-analysis of mandibular distraction osteogenesis: clinical applications and functional outcomes. *Plastic and reconstructive surgery* 2008;**121**(3):p54e–e69.
[95] Hong P, et al. Feeding and mandibular distraction osteogenesis in children with Pierre Robin sequence: a case series of functional outcomes. *International journal of pediatric otorhinolaryngology* 2012;**76**(3):414–8.
[96] Hong P, et al. Mandibular distraction osteogenesis in children with Pierre Robin sequence: impact on health-related quality of life. *International journal of pediatric otorhinolaryngology* 2012;**76**(8):1159–63.
[97] Tahiri Y, et al. The effectiveness of mandibular distraction in improving airway obstruction in the pediatric population. *Plastic and reconstructive surgery* 2014;**133**(3):p352e–e359.
[98] Taylor BA, Brace M, Hong P. Upper airway outcomes following midface distraction osteogenesis: a systematic review. *Journal of plastic reconstructive & aesthetic surgery: JPRAS* 2014;**67**(7):891–9.

Please cite this article in press as: Ngiam J, Cistulli PA. Dental Treatment for Paediatric Obstructive Sleep Apnea. Paediatr. Respir. Rev. (2015), http://dx.doi.org/10.1016/j.prrv.2014.11.002